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Maximal cardiorespiratory responses to one- and two-legged cycling during acute and long-term exposure to 4300 meters altitude

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Summary. During exposure to altitudes greater than about 2200 m, maximal oxygen uptake ($\dot{V}_{O_2 max}$) is immediately diminished in proportion to the reduction in the partial pressure of oxygen in the inspired air. If the exposure lasts longer than a couple of days, an increase in arterial oxygen content (CaO_2), due to a hemoconcentration and an increase in arterial oxygen saturation, occurs. However, there is also a reduction in maximal cardiac output ($\dot{Q}_{max}$) at altitude which offsets the increase in CaO_2 and, therefore, $\dot{V}_{O_2 max}$ does not improve. The purpose of this investigation was to study the contribution of the increase in CaO_2 to the working muscles without the potentially confounding problem of a reduced $\dot{Q}_{max}$. The approach used was to have seven male subjects (aged 17 to 24 years) perform one- and two-legged $\dot{V}_{O_2 max}$ tests on a cycle ergometer at sea level (SL, $PIO_2 = 159$ Torr), after 1 h at 4300 m simulated altitude (SA, $PIO_2 = 94$ Torr) and during two weeks of residence on the summit of Pikes Peak, CO. (PP, 4300 m, $PIO_2 = 94$ Torr). Cardiac output limits maximal performance during two-legged cycling but does not limit performance during one-legged cycling. During the study, CaO_2 changed from 189 ± 3 (mean $\pm SE$) at SL to 161 ± 4 ml·L^{-1} during SA (SL vs. SA, $p < 0.01$) and to 200 ± 6 ml·L^{-1} at PP (SL vs. PP, $p < 0.05$; SA vs. PP, $p < 0.01$). Two-legged $\dot{V}_{O_2 max}$ decreased from 3.64 ± 0.26 L·min^{-1} at SL to 2.70 ± 0.14 L·min^{-1} during SA ($p < 0.01$) to 2.86 ± 0.16 L·min^{-1} at PP ($p < 0.01$). One-legged $\dot{V}_{O_2 max}$ decreased from 2.95 ± 0.22 at SL to 2.25 ± 0.17 L·min^{-1} during SA (SL vs. SA, $p < 0.01$) but improved to 2.66 ± 0.18 L·min^{-1} at PP (SA vs. PP, $p < 0.05$). Since only one-legged $\dot{V}_{O_2 max}$ increased as more oxygen was made available to the working muscles, the altitude-induced reduction in $\dot{Q}_{max}$ can be implicated as being responsible for the reduction in $\dot{V}_{O_2 max}$ during two-legged cycling.

Key words: One-legged cycling — Altitude acclimatization — Cardiac output — Maximal oxygen consumption

Introduction

It is generally agreed that $\dot{V}_{O_2 max}$ is a valid index of the work capacity of an individual because it reflects both the ability of the cardiovascular system to deliver oxygen to the working muscles and the ability of the tissues to utilize oxygen (Astrand and Rodahl 1986). During exposure to high altitude, $\dot{V}_{O_2 max}$ is diminished immediately in an inverse relationship to the elevation beginning at approximately 2200 m (Buskirk 1969; Sutton and Jones 1983). Since $\dot{Q}_{max}$ does not decrease significantly for the first couple of days of exposure to high altitude (Stenberg et al. 1966), the reduction in $\dot{V}_{O_2 max}$ is closely related to the reduction in the CaO_2 (Stenberg et al. 1966; Gleser 1973). During exposures lasting longer than a couple of days, the cause of the sustained reduction in $\dot{V}_{O_2 max}$ is not as firmly established (Alexander et al. 1967; Reeves et al. 1987; Vogel et al. 1967; Wagner et al. 1986.

As the altitude exposure continues and altitude acclimatization occurs, there is an increase in CaO_2 due to both a hemoconcentration (Jung et al. 1971) and an increase in arterial oxygen saturation (Grover et al. 1986). Maximal oxygen uptake, however, does not rise above the levels measured during the acute exposure in proportion to the in-

crease in CaO_2 suggesting that an improvement in $\dot{V}_{O_2max}$ is limited by a reduced $\dot{Q}_{max}$. Although $\dot{Q}_{max}$ is reduced at altitude (Vogel et al. 1974), direct evidence that the reduction actually limits maximal exercise performance is lacking.

If the reduction in $\dot{Q}_{max}$ does limit $\dot{V}_{O_2max}$ at altitude, then increasing $\dot{Q}_{max}$ should increase $\dot{V}_{O_2max}$. However, augmenting $\dot{Q}_{max}$ using methods such as preventing the altitude-induced hemoconcentration to maintain stroke volume (Grover et al. 1976) or by increasing the maximal heart rate with atropine (Hartley et al. 1974) have not been successful in increasing $\dot{V}_{O_2max}$. It may be that the enhanced variable (e.g., stroke volume or heart rate) was offset by a compensatory change in another variable (e.g., CaO_2) making it difficult to determine if restoration of $\dot{Q}_{max}$ can increase $\dot{V}_{O_2max}$.

To examine the contribution of an enhanced CaO_2 without the potentially confounding problem of a reduced $\dot{Q}_{max}$, we determined $\dot{V}_{O_2max}$ during one- and two-legged cycling at sea level and during two weeks of altitude exposure. Cardiac output limits two-legged $\dot{V}_{O_2max}$ at sea level and possibly at altitude (Gleser 1973; Saltin et al. 1968; Vogel et al. 1967), but does not limit one-legged cycling (Gleser 1973). We reasoned that if both one- and two-legged $\dot{V}_{O_2max}$ do not increase as CaO_2 is increased during altitude acclimatization, then other factors such as an impaired ability of the working muscles to fully utilize the available oxygen may be responsible for the reduction in two-legged $\dot{V}_{O_2max}$. If, however, only one-legged $\dot{V}_{O_2max}$ increases with altitude acclimatization, then the altitude-induced reduction in $\dot{Q}_{max}$ can be implicated as being responsible for the reduction in $\dot{V}_{O_2max}$ during two-legged cycling.

Methods

Seven healthy male volunteers served as test subjects. Each gave informed consent and all were highly motivated. Ages ranged from 17 to 24 years (mean: 20.6), heights from 170.2 to 182.9 cm (mean: 173.8) and weights from 63.5 to 80.0 kg (mean: 72.6). The experiment was conducted in the hypobaric environmental chamber at the United States Army Research Institute of Environmental Medicine in Natick, Massachusetts, USA (50 m, $PIO_2 = 159$ Torr), and at the U.S. Army Pikes Peak Laboratory on the summit of Pikes Peak, Colorado (4300 m, $PIO_2 = 94$ Torr). To become familiarized and habituated with the tasks, equipment, and procedures, the subjects were required to frequently practice pedalling on a cycle ergometer with one and or two legs during the first week of the study.

One- and two-legged $\dot{V}_{O_2max}$ tests were performed during two-day periods on five separate occasions: twice at sea level during weeks two and three of the study, once at 4300 m simulated altitude (1-h hypobaric exposure) during week four, and twice at Pikes Peak at the end of weeks seven and eight (the first and second week of the altitude sojourn). On four occasions, during the first day of each two-day period, four subjects completed the two-legged $\dot{V}_{O_2max}$ test and the remaining three subjects completed a one-legged $\dot{V}_{O_2max}$ test. On the following day, the type of $\dot{V}_{O_2max}$ test taken by each of the subjects was reversed. On one occasion at sea level, the order of days the subjects took either the one- or two-legged $\dot{V}_{O_2max}$ test was reversed to determine if there was a test-to-test order effect. The subjects were allowed to continue only in their normal day-to-day activities (primarily sedentary) on the days they were not being tested. Participation in intense physical activity or riding of bicycles was strictly forbidden.

One- and two-legged $\dot{V}_{O_2max}$ were determined using a continuous, incremental cycling protocol on an electrically-braked ergometer (Collins, Inc). All subjects elected to perform the one-legged $\dot{V}_{O_2max}$ test with their right leg. The left leg rested on the middle crossmember of the bike. For all of the one- and two-legged tests, the subjects started pedalling at 50 watts for two minutes followed by an incremental increase of 25 watts every two minutes until they could not continue pedalling. For each of the tests, the subjects were required to pedal at a frequency of 60 rev·min^{-1}. However, during the heaviest workloads of the one-legged $\dot{V}_{O_2max}$ tests, some of the subjects found it necessary to increase the pedalling frequency to 65–70 rev·min^{-1} to maintain the cycling motion. Because the resistance was automatically and immediately reduced to offset the increase in pedalling frequency, the exercise intensity was exactly the same as with 60 rev·min^{-1}. The subjects' feet were secured to the pedals during all $\dot{V}_{O_2max}$ testing.

A Sensormedics Metabolic Measurement Cart Horizon System (MMC; Sensormedics Corp.) was used to collect respiratory metabolic data. The MMC was calibrated with medical grade calibration gases prior to each test. Expired air was channeled from a low resistance valve and tubing into a mixing chamber within the MMC. For each minute, mixed expired gas was sampled from the mixing chamber for 45 seconds alternated with 15 seconds of sampling end-tidal values for oxygen and carbon dioxide directly from the mouthpiece. Analog heart rate signals from a heart rate monitor (IBS, Inc.) were continuously fed to the MMC. Values for heart rate, minute ventilation, oxygen consumption, carbon dioxide production, and respiratory quotient were calculated and printed every 15 seconds. A minute-to-minute summary report which averaged the four 15-second periods of each minute was printed at the conclusion of each test and was subsequently used to provide the information for analysis of the respiratory data.

Hemoglobin and hematocrit were determined prior to each $\dot{V}_{O_2max}$ test from samples obtained from an indwelling catheter placed in an antecubital vein. Plasma volume reduction was calculated using the equation of Dill and Costill (1974). An ear oximeter (Hewlett-Packard, Inc.) was employed periodically throughout the study to determine resting oxygen saturation. Arterial oxygen content (ml·L^{-1}) was calculated as the product of saturation (%) X Hb (g·100 ml^{-1}) X 1.36 ml O_2·g Hb^{-1}.

The data were analyzed using a two-way, repeated-measures analysis of variance (subject X trial). When a statistically significant F-ratio was calculated, differences between the means were tested for significance using Neuman-Keuls post-hoc test. The level of significance was chosen as $p < 0.05$.

Results

There were no significant differences in any of the respiratory or haematological parameters mea-

Table 1. Maximal physiological responses during one- and two-legged exercise at sea level and after 1 hour, 1 week and 2 weeks exposure to 4300 m altitude

Parameter	Sea level	1 h	1 Week	2 Weeks
Workload (watts)				
1 leg	179 ±13	150 ±12**	157 ±12*	171 ±13***
	#	#	#	#
2 legs	268 ±21	207 ±12**	218 ±13**	229 ±13**
$\dot{V}_{O_{2max}}$ (L·min^{-1})				
1 leg	2.95± 0.22	2.25± 0.17**	2.50± 0.19**	2.66± 0.18***
	#	#	#	#
2 legs	3.64± 0.26	2.70± 0.14**	2.75± 0.15**	2.86± 0.16**
$\dot{V}_{CO_{2max}}$ (L·min^{-1})				
1 leg	3.41± 0.23	3.02± 0.23*	3.03± 0.20*	3.21± 0.17
	#			
2 legs	4.28± 0.28	3.42± 0.17**	3.23± 0.16**	3.26± 0.22**
R ($\dot{V}_{CO_{2max}} \cdot \dot{V}_{O_{2max}}^{-1}$)				
1 leg	1.16± 0.00	1.34± 0.03*	1.22± 0.02***	1.21± 0.02***
		#	#	
2 legs	1.18± 0.02	1.28± 0.02*	1.18± 0.02***	1.19± 0.01***
Heart rate (beats·min^{-1})				
1 leg	173 ± 4	165 ± 4	172 ± 4	172 ± 2
	#	#		#
2 legs	188 ± 2	177 ± 2*	176 ± 2*	176 ± 2*
Ventilation (L·min^{-1})				
1 leg	124 ±13	132 ±15	179 ±17* ***	207 ±15** ***
	#			#
2 legs	149 ±15	150 ±15	178 ±14* ***	192 ±14* ***

Values are means ±SE

 # The 1-legged value is significantly different than the 2-legged value ($p < 0.01$)

 * Significantly different from sea level ($p < 0.05$)

 ** Significantly different from sea level ($p < 0.01$)

 *** Significantly different from the first hour of exposure ($p < 0.05$)

sured during rest or during either exercise protocol for the two testing sessions at sea level. It was also determined that the test-to-test sequence of $\dot{V}_{O_{2max}}$ testing had no influence on any of the results obtained. Therefore, the values collected on the latter test session at sea level were used as the sea-level baseline values.

The maximal physiological responses to one- and two-legged exercise are presented in Table 1. Two-legged $\dot{V}_{O_{2max}}$ was reduced 26% during the first hour of exposure, 25% during the first week, and 22% during the second week at Pikes Peak. Thus, regardless of the length of exposure, two-legged $\dot{V}_{O_{2max}}$ was significantly reduced from sea-level values as illustrated in Fig. 1. Conversely, one-legged $\dot{V}_{O_{2max}}$ was decreased 24% in the first hour of exposure but gradually increased 18% by the second week at Pikes Peak to a $\dot{V}_{O_{2max}}$ value that was not significantly different from sea level. At sea level and during the first hour of exposure, the one-legged $\dot{V}_{O_{2max}}$ values were 81% and 83% of the two-legged $\dot{V}_{O_{2max}}$ values, respectively.

At Pikes Peak, the one- to two-legged $\dot{V}_{O_{2max}}$ ratios were 91% during week one and 93% during week two.
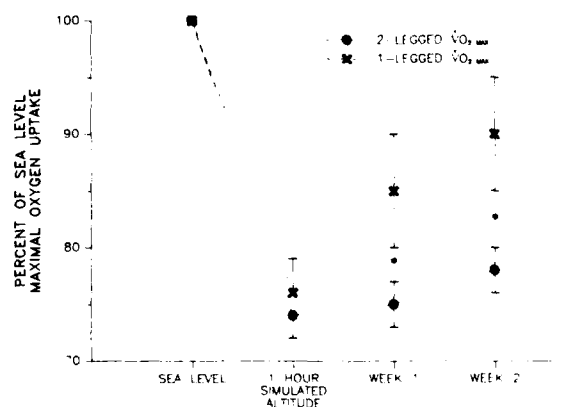

Fig. 1. Percentage of sea level maximal oxygen uptake for one- and two-legged cycling. Values are means ±SE. * Significant difference between one- and two-legged $\dot{V}_{O_{2max}}$ values ($p < 0.01$)

Table 2. Hemoglobin, hematocrit, plasma volume reduction, and arterial blood saturation at sea level and after 1 h, 1 week, and 2 weeks of exposure to 4300 m altitude

Parameter	Sea level	1 h	1 Week	2 Weeks
Hemoglobin (g·100 ml^{-1})	14.5±0.2	14.1±0.2	15.9±0.3** ***	16.6±0.2** ***
Hematocrit (%)	42.1±0.4	41.6±0.6	46.0±1.0** ***	48.1±0.7** ***
Plasma Volume	—	4.6±2.4	−12.7±3.2* ***	−21.2±2.2** ***
(% change from sea level)				
O_2 Saturation (%)	96 ±1	84 ±1*	87 ±1* ***	88 ±1* ***
O_2 Content (ml·L^{-1})	189 ±3	161 ±4**	186 ±7***	200 ±6* ***

Values are means ±SE
*   Significantly different from sea level ($p < 0.05$)
**   Significantly different from sea level ($p < 0.01$)
*** Significantly different from the first hour of exposure ($p < 0.01$)

Maximal heart rates during two-legged cycling during the one-hour exposure and at Pikes Peak were reduced from sea level by 11 beats·min^{-1} and 12 beats·min^{-1}, respectively ($p < 0.05$). Maximal heart rates during one-legged cycling at altitude were not statistically different from sea level. At sea level and at altitude, the maximal heart rates for one-legged cycling were always less than the maximal heart rates for two-legged cycling.

Maximal minute ventilation increased for both one- and two-legged cycling during the sojourn at Pikes Peak. Ventilation was greater for two-legged cycling than for one-legged cycling only at sea level ($p < 0.05$). During the one-hour exposure and through the first week at Pikes Peak, ventilations for one- and two-legged cycling were not statistically different from each other. However, during the second week at Pikes Peak, ventilations during one-legged exercise were 8% higher ($p < 0.01$) than the ventilations during two-legged cycling.

Table 2 presents the values for hemoglobin, hematocrit, plasma volume, oxygen saturation, and CaO_2. From sea level to the one-hour exposure in the altitude chamber, hemoglobin, hematocrit and plasma volume were not altered while oxygen saturation and CaO_2 were reduced 12.5% and 14.8%, respectively. During the Pikes Peak sojourn, however, hemoglobin and hematocrit were increased significantly above sea level baseline values ($p < 0.01$). From these values, it was estimated that plasma volume was reduced by 21.2% in the second week. Also, in the second week of the sojourn, oxygen saturation increased 5% above the values obtained during the one-hour exposure (84% to 88%). Because of the increases in oxygen saturation and hemoglobin at Pikes Peak, CaO_2 increased from 161 ml·L^{-1} during the one-hour exposure to 186 ml·L^{-1} (+15.5%) during week one and to 200 ml·L^{-1} (+24.2%) during week two.

Discussion

This study was designed to eliminate as many extraneous factors as possible so that a true comparison could be made between the maximal responses of one- and two-legged cycling at sea level and at altitude. To that end, the test subjects, equipment, diet, times of testing, testing conditions and cycling protocols were identical for the one- and two-legged $\dot{V}_{O_2 max}$ tests. Also, a minimum of one week separated consecutive testing periods so that an exercise "training effect" would not occur.

In this study, one-legged $\dot{V}_{O_2 max}$ was approximately 82% of the two-legged value at sea level and during the first hour of altitude exposure. This relationship is similar to values previously reported at sea level (Gleser 1973; Neary and Wenger 1986; Davies and Sargeant 1975) and during acute hypoxia (Gleser 1973). The 25% reduction from sea level to the first hour of exposure for one- and two-legged $\dot{V}_{O_2 max}$ is also similar to the findings of previous investigations using similar altitudes or levels of hypoxia (Buskirk 1969; Gleser 1973). In contrast to some studies (Gleser 1973; Vogel et al. 1974; Vogel and Gleser 1972) but not all (Ekblom et al. 1975), the reductions in one- and two-legged $\dot{V}_{O_2 max}$ from sea level to the one-hour exposure found in the present study were not entirely due to the 14.8% reduction in CaO_2. The reductions in the maximal heart rate values obtained during one-legged (−8 beats·min^{-1}; ns) and two-legged (−11 beats·min^{-1}; $p < 0.05$) cycling were also contributory.

As the altitude exposure continued, two-legged $\dot{V}_{O_2 max}$ values did not increase significantly above the values obtained during the first hour of exposure and thus remained below sea-level values despite a 24.2% increase in CaO_2 (161 $ml \cdot L^{-1}$ to 200 $ml \cdot L^{-1}$). These observations have been reported on numerous occasions (Buskirk 1969; Vogel et al. 1967). What has not been reported previously is that $\dot{V}_{O_2 max}$ during one-legged cycling improved 11.1% and 18.2% from one hour of exposure to the first and second weeks of altitude exposure, respectively. Furthermore, the improvement in one-legged $\dot{V}_{O_2 max}$ was so pronounced that the value obtained during week two did not differ from the sea level value.

It is well established from previous studies that $\dot{Q}_{max}$ is reduced after the first few days at altitudes greater than 3000 m (Alexander et al. 1967; Klausen 1966; Vogel et al. 1967). The 21.2% reduction in plasma volume calculated in the present study is consistent with a reduction in $\dot{Q}_{max}$. It has also been shown that $\dot{Q}_{max}$ during one-legged cycling is 75% to 87% of two-legged cycling and is clearly not a limiting factor to one-legged maximal performance at sea level (Gleser 1973; Davies and Sargeant 1974; Stamford et al. 1978) or during acute hypoxia (Gleser 1973). Throughout the present study, maximal heart rates during one-legged cycling were not as high as the maximal heart rates during two-legged cycling strongly suggesting that $\dot{Q}_{max}$ does not limit one-legged maximal exercise performance even after two weeks of altitude exposure. One-legged $\dot{V}_{O_2 max}$ appears to be limited by the ability of the muscle vasculature to accept the high blood flow (Gleser 1973; Stamford et al. 1978).

During normoxia, maximal two-legged cycling causes a greater arterial desaturation than one-legged cycling because of the limits imposed by $\dot{Q}_{max}$ (Davies and Sargeant 1974; Stamford et al. 1978). Consequently, when air enriched with oxygen is breathed, two-legged but not one-legged $\dot{V}_{O_2 max}$ is increased (Davies and Sargeant 1974). In the present study, in a similar manner, there was an increase in availability of oxygen to the working muscles due to an increase in CaO_2. After the first week of exposure, during two-legged maximal exercise, the increases in oxygen availability and utilization were offset by the altitude-induced reduction in $\dot{Q}_{max}$ so that the $\dot{V}_{O_2 max}$ value remained at a level similar to that measured during the one-hour chamber exposure (Grover 1986).

During one-legged cycling, $\dot{V}_{O_2 max}$ increased as the altitude exposure lengthened. Why would an increase in oxygen availability cause a rise in one-legged $\dot{V}_{O_2 max}$ at altitude when it does not affect maximal performance at sea level? It is not likely that this increase was related to arterial desaturation because $\dot{Q}_{max}$ during one-legged cycling was not as high as with two-legged cycling. It is also not likely that there was a greater redistribution of flow to the working muscles due to an exercise "training effect" since the number of $\dot{V}_{O_2 max}$ testing sessions was limited to a maximum of once a week with the subjects remaining relatively sedentary on the days they were not being tested. Furthermore, one-legged $\dot{V}_{O_2 max}$ measured during the first week at Pikes Peak was improved even through the test subjects had not performed any cycling ($\dot{V}_{O_2 max}$ testing or recreational) during the three weeks prior to this $\dot{V}_{O_2 max}$ test. One possible explanation for the improvement in one-legged $\dot{V}_{O_2 max}$ at altitude may be that structural and/or metabolic adaptations in the working muscles or an increase in diffusion capacity from the capillary to the tissue mitochondria occurred, favoring a more complete extraction and utilization of oxygen (Sutton et al. 1988). Direct evidence of these changes occurring, however, was beyond the scope of this study.

In conclusion, two-legged $\dot{V}_{O_2 max}$ is reduced at altitude. The component of systemic oxygen transport responsible for the early reduction in $\dot{V}_{O_2 max}$ is the low CaO_2, whereas decreased $\dot{Q}_{max}$ is the reason for the persistence of the impairment after several days at altitude.

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